

A Prospective Analysis of Trauma Exposure: The Mediating Role of PTSD Symptomatology

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Trauma exposure has been associated with increased risk of exposure to additional traumatic events. Reactions to trauma exposure, specifically PTSD symptomatology, may mediate the link between trauma exposure and later traumatic events. Data from a longitudinal sample of Gulf War veterans ($N = 2,949$) were analyzed using a series of regression models. Higher levels of combat exposure were related to increased reports of PTSD symptomatology immediately upon return as well as increased reports of traumatic events in the 2 years following the Gulf War. PTSD symptomatology partially mediated the link between combat exposure and later trauma. Symptom clusters were also analyzed separately as potential mediators. Implications for the treatment of PTSD and prevention of exposure to multiple traumas are discussed.

KEY WORDS: PTSD; Gulf War veterans; trauma exposure; retraumatization.

The long-term ramifications of exposure to trauma are not well understood. Evidence suggests that one consequence of trauma exposure may be an increased risk of exposure to additional trauma (Hanson, Kilpatrick, Falsetti, & Resnick, 1995). Indeed, results from a large-scale longitudinal survey suggest that women who have a history of rape or aggravated assault are approximately twice as likely to experience another assault than those without such a history (Kilpatrick, Resnick, Saunders, & Best, 1998). Very little is known, however, about the mechanisms through which exposure to trauma may result in increased risk of exposure to future trauma. One possible mechanism through which this vulnerability operates is the individual's response to the trauma. The experience of posttraumatic stress disorder (PTSD) symptoms is quite common following trauma exposure. Several longitudinal studies report that 30% of individuals meet full criteria

for PTSD 1 month following trauma exposure (see, e.g., Shalev et al., 1998), although rates as high as 94% have been reported among women assessed within 1 month of a sexual assault (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). It may be that the experience of PTSD symptoms (both acutely and across time) uniquely contributes to the development of a vulnerability to future exposure, for example, through deficits in self-protective behaviors. To explore this possibility, this study examined prospectively whether exposure to combat during the Gulf War was associated with increased risk of exposure to traumatic events in the 2 years following the Gulf War. In addition, PTSD symptoms, as a whole and separately by symptom cluster, were examined as a potential mediator of the relationship between combat exposure and subsequent exposure to traumatic events.

The link between trauma exposure and increased risk for additional trauma exposure has been documented most extensively with regard to sexual trauma, particularly childhood sexual assault (see Messman & Long, 1996, for a review). The majority of studies in this area, however, are retrospective. A handful of prospective studies (Collins, 1998; Gidycz, Coble, Latham, & Layman, 1993; Gidycz, Hanson, & Layman, 1995; Kilpatrick et al., 1998) have

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found evidence for a link between trauma and risk for future trauma exposure, particularly with regard to sexual victimization. Defining trauma exposure more broadly (e.g., motor vehicle accident, physical assault, witnessing severe injury or death), Breslau, Davis, and Andreski (1995) followed a large random sample of young adult members of a Detroit health maintenance organization for 3 years, and reported that people endorsing a history of trauma exposure at Time 1 were nearly twice as likely as those not reporting a trauma history to be exposed to trauma during the follow-up period. There is little empirical data, however, on the link between combat exposure, specifically, and risk for subsequent exposure to trauma. One recent cross-sectional retrospective study (King, King, Fairbank, Keane, & Adams, 1998) demonstrated a link between combat exposure during Vietnam and later exposure to stressful life events among a large sample of Vietnam veterans.

One mechanism through which trauma exposure might increase an individual's risk for subsequent exposure to trauma is via the individual's response to the stressor, specifically, whether an individual experiences PTSD symptoms following trauma exposure. PTSD has been consistently documented as a consequence of trauma exposure in civilians (see, e.g., Aciermo, Kilpatrick, & Resnick, 1999) and in veterans following combat exposure (see, e.g., Kulka et al., 1988). To the authors' knowledge, however, only one study (Sandberg, Matorin, & Lynn, 1999) has examined the relation between PTSD and risk for trauma exposure in a prospective manner. Using a sample of college women, Sandberg et al. (1999) found that PTSD symptomatology was not significantly related to subsequent trauma and thus did not meet the conditions for mediation. These investigators did find that prior victimization was more strongly associated with subsequent victimization among those with higher levels of symptomatology, suggesting that PTSD symptomatology could act as a mediator for women with higher levels of the disorder.

This study represents an important first step in exploring the potential role of PTSD as a mediator of risk for trauma exposure. This longitudinal study tests whether PTSD may act as a mediator between combat exposure and later trauma exposure among Gulf War veterans. It was hypothesized that higher combat exposure during the Gulf War would predict both higher PTSD symptomatology reported upon homecoming and greater exposure to traumatic life events in the approximately 2 years following the Gulf War. In addition, it was hypothesized that higher levels of PTSD symptomatology would be related to greater exposure to traumatic life events after the Gulf War, and that PTSD symptomatology would mediate the

relationship between combat exposure and later traumatic events as demonstrated by a reduced relationship between combat exposure and later traumatic events when PTSD symptomatology is controlled in the model. Finally, the three PTSD symptom clusters (i.e., reexperiencing, avoidance and numbing, and hyperarousal) were examined separately to explore the relative influence of the symptom clusters in the mediational model.

Method

Participants

Data for this study were obtained from a longitudinal investigation of Gulf War veterans, interviewed initially in 1991 and again in 1992–93. On both occasions, informed consent was obtained after the study was described to the respondents. At Time 1, 2,949 Army personnel at Ft. Devens, Massachusetts, completed a 45-min paper and pencil questionnaire within 5 days of their return to the United States from the Gulf region. Approximately 60% of those deployed from Ft. Devens to the Gulf region were surveyed; those not surveyed were typically unavailable due to general administrative purposes (e.g., outprocessing). At Time 1 (1991), participants included 2,702 men and 240 women. The majority of the cohort (72%) was called from the Reserves or National Guard (as opposed to active duty), and most were enlisted personnel (92%) as opposed to officers. The cohort was predominantly White (82%) with 9% Black, 4% Hispanic, and 5% designated as "other." The mean age was 30 years with an average education level of just over 13 years. Men and women at Time 1 significantly differed on age (i.e., women were younger), education (i.e., women were more educated), ethnicity (i.e., women were more likely to be members of an ethnic minority group), and marital status (i.e., women were less likely to be currently married), all $p < .05$. Of the 2,313 participants who completed the Time 2 (1992–93) survey (response rate of 78%), 922 (40%) completed the survey during face-to-face unit meetings. Those unavailable at unit meetings were contacted by mail and 1,086 (47%) completed the survey in this manner. The remaining 304 participants (13%) did not return mailed surveys and were contacted and completed surveys during phone interviews. Comparison of the demographic characteristics of reinterviewed and nonreinterviewed respondents on a number of Time 1 variables revealed no significant differences in gender, education level, marital status, military rank, prior combat experience, Gulf War combat exposure, and PTSD. However older respondents, White respondents, and

respondents called from the Reserve or National Guard as compared to active duty were more likely to be reinterviewed (see Wolfe, Erickson, Sharkansky, King, & King, 1999, for a complete description).

Measures

At both time points, study participants completed a battery of self-report measures assessing background and demographic information, their experiences in the Gulf region, and psychological outcomes, including PTSD.

Covariates

Gender, age (in years), education level (in years), and marital status (married vs. not married) measured at Time 1 were included as possible covariates due to their significant association with PTSD in the National Comorbidity Survey (Kessler et al., 1999).

Gulf War Combat Exposure

The Laufer Combat Scale (Gallop, Laufer, & Yager, 1981) was used at Time 1 to assess combat exposure during the Gulf War. In this study, the Laufer Scale was augmented with items that pertained to distinctive Gulf War experiences such as being on alert for SCUD or biochemical attack (see Wolfe et al., 1999). The augmented measure contained 33 self-report items and used a 3-point Likert response format (0 = *never*, 1 = *once or twice*, and 2 = *three or more times*). The 33 items were summed to create a total Gulf War combat exposure scale (coefficient $\alpha = .73$).

PTSD Symptomatology

PTSD symptomatology was evaluated at Time 1 using the original Mississippi Scale for Combat-Related PTSD (Keane, Caddell, & Taylor, 1988) modified slightly for Gulf War personnel (see Wolfe et al., 1999, for more information). The measure has demonstrated excellent specificity and sensitivity with clinical diagnoses of PTSD (Keane et al., 1988). A rational approach was used in this study to identify a subset of Mississippi items that most closely mapped onto the 17 PTSD symptoms in the *DSM-IV* (*Diagnostic and statistical manual of mental disorder*, 4th ed.; American Psychiatric Association, 1994). Four PhD-level clinicians identified five items for each of the three symptom clusters (reexperiencing, avoidance and numbing, and hyperarousal) as the most saturated with content (see Erickson et al., 2000, for additional

details on construction of this subset of items).⁴ Internal consistency reliability (coefficient α) for the final set of 15 items was .83. The subset of 15 items correlated .89 with the full set of items. The possible range of scores was 1–5 with higher scores indicating greater symptomatology.

Traumatic Life Events at Time 2

Based on the Traumatic Stress Schedule (Norris, 1990), traumatic life events (TLE) at Time 2 was measured as a count of four life events that may have occurred since the Time 1 interview (the number endorsing each event are in parentheses): (1) very serious accident, illness, or medical procedure involving yourself ($n = 313$), (2) loss of your home or property due to fire, flood, or other natural or man-made disaster ($n = 41$), (3) an assault (including robbery, mugging, or rape) of yourself ($n = 64$), and (4) an event where you saw someone you didn't know badly hurt or violently killed ($n = 272$). Participants responded "yes" or "no" to each of the four events and rated the impact of each event from 1 (*None*) to 5 (*Extreme*). Only events with an impact rating of 2 or higher were included in the overall count. To minimize effects of nonnormality (skew = 2.14, kurtosis = 5.55) on the analyses, a rank normal transformed total score was computed and used in all analyses ($M = 0.30$, $SD = 0.57$, Range = 0–4, for nontransformed variable, $M = 0.00$, $SD = 0.70$ for transformed variable).

Overview of Analyses

PTSD symptomatology was examined as a possible mediator of the relation between combat exposure and subsequent exposure to trauma. Prior research (King &

⁴Reexperiencing items: (1) If something happens that reminds me of Operation Desert Storm, I become very distressed and upset, (2) I have nightmares of experiences in Southwest Asia that really happened, (3) Being in certain situations makes me feel as though I am back in Southwest Asia, (4) At times, I suddenly act or feel as though something that happened in Operation Desert Storm were happening all over again, (5) If something happens that reminds me of Operation Desert Storm, I get so anxious and panicky that my heart pounds hard; I have trouble getting my breath, I sweat, tremble, or shake; or feel dizzy, tingly or faint (Cronbach's $\alpha = .78$). Hyperarousal items: (1) I have trouble concentrating on tasks, (2) I fall asleep easily at night, (3) Unexpected noises make me jump, (4) I lose my cool and explode over minor everyday things, (5) I feel "superalert" or "on guard" much of the time (Cronbach's $\alpha = .58$). Numbing items: (1) Before my participation in Operation Desert Storm, I had more close friends than I have now, (2) It seems as if I have no feelings, (3) I still enjoy doing many things that I used to enjoy, (4) I try to stay away from anything that will remind me of things which happened while I was in Southwest Asia, (5) I am not able to remember some important things that happened in Southwest Asia (Cronbach's $\alpha = .54$).

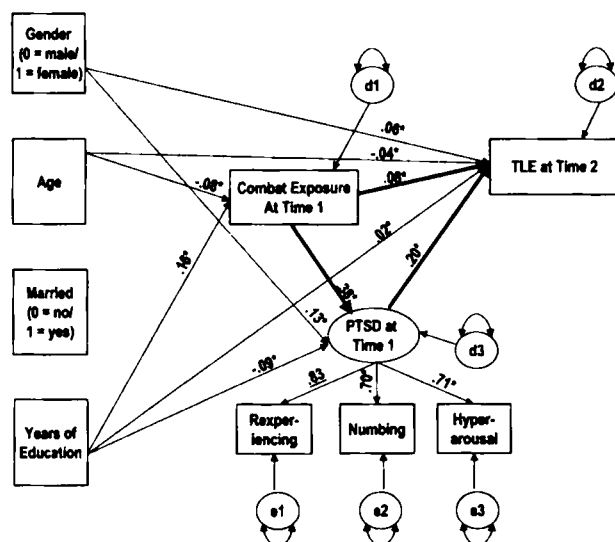


Fig. 1. Final model examining PTSD symptoms at Time 1 as a mediator of the relation between combat exposure at Time 1 and traumatic life events (TLE) at Time 2. All paths significant at $p < .05$. Covariates and nonsignificant paths are not depicted in the figure. β for covariates are as follows: gender with age (.07), marital status (.14), and education (.07); age with marital status (.37), and education (.16); and marital status with education (.02).

King, 1994) has indicated that a unidimensional, latent variable of PTSD symptoms can be estimated with the Mississippi Scale for Combat-Related PTSD. In the current analyses, three scale scores pertaining to the three *DSM-IV* symptom clusters (reexperiencing, avoidance and numbing, and hyperarousal) were calculated by averaging across their respective items. These scale scores were then used as indicators for a latent variable of PTSD. Combat exposure (collected at Time 1) and traumatic life events (TLE) since Time 1 (collected at Time 2) were modeled as manifest indicators defined as scale sum scores (Fig. 1).

According to Baron and Kenny (1986), four conditions must be met to establish mediation. First, the predictor must be significantly related to the outcome. To test this, TLE at Time 2 was regressed on combat exposure at Time 1. Second, the predictor must be significantly related to the potential mediator. To test this, PTSD was regressed on combat exposure. Third, the potential mediator must be significantly related to the outcome. To test this, TLE was regressed on PTSD. Fourth and finally, the effect of the predictor on the outcome must be significantly reduced when controlling for the effects of the mediator. To test this, TLE was regressed on both combat exposure and PTSD. Full mediation is indicated if the effect from combat exposure to TLE is zero and partial mediation is indicated if the relation is significantly reduced (relative to the regression of TLE solely on combat exposure), albeit not to zero. With partial mediation, one measure of the magnitude of effect is the proportion of the total effect mediated (MacKinnon, Warsi, & Dwyer, 1995).

All regressions were estimated using the AMOS program (Arbuckle & Wothke, 1999). This program has two advantages over a traditional multiple regression programs. First, it allowed for the use of a latent variable to measure PTSD and, second, it used maximum likelihood methods to deal with missing data at Time 2.

Results

Descriptive Statistics

Means, standard deviations, and intercorrelations for age, gender, marital status, education, combat exposure, TLE at Time 2, and PTSD symptoms (as indexed by Mississippi scale total score) are presented in Table 1. Age, gender, marital status, and education were examined for

Table 1. Means, Standard Deviations, and Intercorrelations for Study Variables

	Correlations									<i>M (SD)</i>	<i>N</i>
	1	2	3	4	5	6	7	8	9		
1. Age										30.16 (8.42)	2,880
2. Gender (0 = Male/1 = Female)	-.07***									0.08 (0.27)	2,942
3. Married (0 = No/1 = Yes)	.37***	-.14***								0.57 (0.49)	2,931
4. Years of education	.16***	.07***	.03							13.17 (1.80)	2,934
5. Number of TLE ^a	-.07***	.09***	-.04	.03						0.06 (0.70)	2,280
6. Combat exposure	-.05**	-.00	-.02	.15***	.16***					6.97 (4.96)	2,936
7. PTSD symptoms ^b	-.08***	.14***	-.06***	-.08***	.20***	.26***				61.90 (13.35)	2,913
8. Reexperiencing	-.07***	.13***	-.04*	.01	.20***	.34**	.72***			1.35 (0.48)	2,918
9. Avoidance and numbing	-.07***	.05**	-.06***	-.03	.17***	.21***	.76***	.58***		1.50 (0.49)	2,927
10. Hyperarousal	-.06***	.09***	-.04*	-.07***	.17***	.25***	.76***	.59***	.51***	2.14 (0.61)	2,927

^aTraumatic life events (TLE) is rank normal transformed.

^bPTSD symptoms computed as a sum of Mississippi scale items.

* $p < .05$. ** $p < .01$. *** $p < .001$.

inclusion as possible covariates. As can be seen in Table 1, the demographic variables were significantly correlated with combat exposure, TLE at Time 2, and/or PTSD symptoms, and were controlled in the models described below.

PTSD Modeled as a Latent Variable

PTSD was modeled as a latent variable with three manifest indicators (pertaining to reexperiencing, avoidance and numbing, and hyperarousal symptoms), where each indicator was an average score of the five items of the Mississippi scale determined to best measure the appropriate *DSM-IV* symptoms for that cluster. The factor loadings were quite good, with standardized coefficients of .82 for reexperiencing, .71 for avoidance and numbing, and .72 for hyperarousal.

Mediation Analyses

The first three conditions of the mediation model were met. Specifically, controlling for the effects of the covariates, (1) combat exposure at Time 1 was positively and significantly associated with traumatic life events at Time 2 ($\beta = .16, p < .05$); (2) combat exposure was significantly and positively associated with PTSD symptoms ($\beta = .38, p < .05$); and (3) PTSD was positively and significantly associated with traumatic life events ($\beta = .23, p < .05$).

To test the fourth and final condition, traumatic life events were regressed on both PTSD and combat exposure, again controlling for the effects of the four covariates. Against the backdrop of significant effects for age, education level, and gender, PTSD was significantly related to traumatic life events ($\beta = .20, p < .05$), and, controlling for this effect, the direct effect of combat exposure on traumatic life events was .08 ($p < .05$). Although still significant, the direct effect of combat exposure on later traumatic events was reduced by 48% (from .16 to .08) when controlling for the effect of PTSD symptomatology. Moreover, using an unbiased estimate of the variance, the mediated effect (as measured by the product of the standardized regression coefficients from combat exposure to PTSD and PTSD to later traumatic events) was statistically significant (see MacKinnon et al., 1995, for a complete description of this technique). Given that PTSD was modeled as a latent variable, the associated regression model had a positive degree of freedom and the associated model fit was quite good, $\chi^2(12) = 72.17$, root mean square error of approximation = .041, Tucker–Lewis coefficient = .995, comparative fit index = .999. Together, these findings suggest that PTSD symptomatology *partially* mediates the link between combat exposure and later traumatic

events. A significant direct effect of combat exposure on later traumatic life events remained, however, and was not accounted for by PTSD symptomatology.

In addition to examining PTSD symptoms overall as a possible mediator of the relation between combat exposure and traumatic life events, these analyses were repeated separately for each of the three component symptom clusters of PTSD, namely reexperiencing, avoidance and numbing, and hyperarousal. Once again using the four conditions of mediation outlined above, results suggest that the effects seen with PTSD overall were also evident in each of the symptom clusters, albeit to a somewhat smaller degree. Specifically, reexperiencing symptoms were found to partially mediate the relation between combat exposure and traumatic life events, with the overall effect of combat exposure reduced from a standardized coefficient of .16 to a standardized coefficient of .11 with the addition of reexperiencing symptoms. This represents a 33% reduction, and, using the criteria outlined above, is statistically significant. Avoidance and numbing as well as hyperarousal were also found to partially mediate the effects of combat exposure on traumatic life events. Results for avoidance and numbing as well as hyperarousal were more modest, with each reducing the effect from .16 to .13 (an approximately 20% reduction, a statistically significant effect). Thus, all three clusters significantly mediated the relationship between combat exposure and traumatic life events. Although the reexperiencing cluster appeared to account for a larger proportion of variance than the other two clusters, it is important to note that the present analyses do not allow us to conclude that reexperiencing symptoms accounted for *significantly* more variance than either of the other two clusters.

Sensitivity Analyses

Two additional analyses were conducted to examine the robustness of the present findings to several alternative measurement constructions. First, the model was reestimated using a revised version of the TLE measure that included all events endorsed regardless of the impact rating. That is, if an individual reported being assaulted, but indicated that this had no impact on his/her life, we included this in the revised TLE count. Not surprisingly, the pattern of results was the same although weaker. Specifically, in the final mediational model, the path from combat exposure to the revised TLE variable was reduced from .18 to .11, a reduction of 38% as compared to the 48% obtained above. Second, we conducted the mediation analyses using the sum score for the Mississippi instead of the latent PTSD construct. Again, we found that the pattern of results was the same although weaker. We found

a reduction of 31% from .16 to .11 in the final mediational model. Given that latent variables are more reliable than manifest variables, this attenuation of results is to be expected. Thus, the present analyses suggest that the present pattern of results is generally robust to several alternative measurement constructions.

Discussion

This study demonstrated that trauma exposure prospectively predicted increased risk for exposure to subsequent trauma. Specifically, combat exposure during the Gulf War was associated with an increased risk of experiencing traumatic events in the approximately 2-year period following participants' return to the United States. Demonstrating a prospective link between combat exposure and increased risk for subsequent traumatic events extends the finding that trauma increases risk of subsequent trauma exposure beyond the area of sexual victimization. Although previous research examining the risk of subsequent trauma exposure has generally examined this link among women, this study used a predominantly male sample. Thus, this study suggests that increased risk of subsequent trauma exposure following trauma exposure is not limited to the domain of sexual trauma, nor to women.

This study also demonstrated that, among Gulf War veterans, PTSD symptomatology partially mediates the relationship between trauma exposure and subsequent trauma exposure. Thus, it appears that at least one specific response to trauma, PTSD symptomatology, may play a role in conferring risk for subsequent trauma exposure. In this study, PTSD symptomatology mediated 48% of the total effect of combat exposure on risk for subsequent exposure to trauma. This study provided preliminary evidence that reexperiencing symptoms may confer potentially more risk of retraumatization than the avoidance and numbing, and hyperarousal symptom clusters. These results must be interpreted with caution, however, given that this hypothesis is based on the relatively larger path coefficient for reexperiencing symptoms rather than a statistical test comparing the predictive ability of the three clusters.

What are some possible mechanisms through which PTSD symptoms would increase risk of additional trauma exposure? It may be that individuals with PTSD experience increased risk of additional trauma exposure as a result of a deficit in self-protective behaviors. This deficit may occur through at least three, nonexclusive pathways. First, PTSD may result in an increase in behavior (e.g., substance use) that magnifies risk for trauma. Evidence suggests that individuals with PTSD are at increased risk

of substance use (Kilpatrick et al., 2000) and that substance use, in turn, has been linked to increased risk of trauma exposure (see, e.g., Kilpatrick, Acierno, Resnick, Saunders, & Best, 1997). It has been theorized that substance use increases risk of trauma exposure due to factors such as impaired ability to detect assailants, increased attractiveness to assailants as a vulnerable target, or risk associated with lifestyles related to substance use (Kilpatrick et al., 1997). Thus, PTSD may set in motion behaviors, such as substance use, that increase risk of trauma exposure.

Second, PTSD may impair information processing, resulting in deficits in accurately recognizing risk. Accurately recognizing risk is cognitively demanding and requires processing of multiple inputs. PTSD symptoms, however, may decrease available cognitive resources. For example, difficulty maintaining concentration is a commonly endorsed symptom of PTSD (McFarlane, 1988). In addition, McNally (1998) has argued that the crucial dysfunction in PTSD may be a failure to inhibit trauma information once it is accessed rather than enhanced accessibility of trauma-relevant material. This difficulty in inhibiting trauma-relevant information is consonant with the common phenomenon of intrusive recollections in PTSD. Thus, individuals with PTSD are operating with an increased cognitive load, and this could decrease an individual's self-protective behavior through a failure to accurately and efficiently process risk-relevant information as it occurs in the environment.

Finally, PTSD may impair self-protective behavior by negatively impacting an individual's ability to disengage from risky situations. The arousal symptoms of PTSD (particularly hypervigilance) may lead to risk perception that has high sensitivity but low specificity. That is, individuals with PTSD may come to overperceive risk and danger in the world, resulting in false alarms. Multiple false alarms may lead to a disconnect between emotional arousal and goal-directed action (Van der Kolk, 1996) such that emotional arousal (e.g., feelings of fear or threat) fails to cue the self-protective behavior necessary to avoid a risky situation. Thus we argue that individuals with PTSD, despite their increased attention to threat and danger, may show deficits in risk disengagement (i.e., active avoidance) due to impaired information processing and a disconnection between emotional arousal and self-protective action. Clearly, deficits in risk disengagement may increase risk of additional trauma exposure.

The finding that PTSD symptomatology contributes to the link between trauma exposure and increased risk for additional trauma exposure has important implications for prevention of trauma exposure. Specifically, if an individual is exposed to trauma and develops PTSD

symptomatology, therapeutic intervention to minimize symptoms may also reduce an individual's risk of exposure to future trauma. Treatment of PTSD symptoms may therefore exert a significant role in breaking a cycle of multiple traumatizations. A greater understanding of the mechanisms by which PTSD impacts risk for re-exposure will improve the quality of posttrauma treatments. For example, if PTSD mediates risk for re-exposure through a deficit in self-protective behaviors, these behaviors could be specifically targeted in order to reduce risk. Although a discussion of self-protective behaviors necessarily conveys individual responsibility for trauma exposure, it should be noted that we believe that the responsibility for traumatic events perpetrated by others (e.g., assault) lies fully with those who perpetrate such acts. That said, the potential individual and societal benefit of identifying and targeting factors that may increase risk for trauma exposure is enormous.

Although PTSD symptomatology functioned as a mediator in this study, it did not fully mediate the relationship between combat exposure and subsequent trauma, suggesting that other factors may also account for increased risk of multiple trauma exposure. For example, dissociation has been theorized as a risk factor for subsequent trauma exposure perhaps due to decreased awareness of surroundings or increased attractiveness as a target because of a confused or distracted appearance (Cloitre, Scarvalone, & Difede, 1997). Depression may also serve to increase vulnerability to future trauma exposure. Another potential explanation for the finding that earlier trauma exposure increases risk for subsequent trauma exposure involves the role of an untested, or "third" variable, that increases risk for both earlier and later trauma exposures. Potential third variable explanations for this phenomenon could include demographic and genetic/biological factors.

There are some limitations to this study. For example, the lack of preexposure assessments of potential explanatory variables limits the ability to rule out potential third variable explanations. In addition, we do not have information on participants' trauma history prior to the Gulf War. When possible, future studies should incorporate assessments of preexposure functioning including a complete trauma history. As is common with wartime studies, information on exposure to combat during the Gulf War was obtained retrospectively and therefore may be biased. Combat exposure served as the sole stressor assessed at Time 1; future studies might include a broader range of stressors (e.g., sexual and nonsexual assault, motor vehicle accidents) to examine potential differential effects on risk for additional trauma exposure (e.g., rape may confer greater risk for additional trauma exposure than

motor vehicle accidents). Further, the measurement of life events in this study did not allow for examination of the impact of one versus multiple incidents of trauma (e.g., participants indicated if they had been in a motor vehicle accident but not how many accidents they experienced). Another measurement issue involves the use of a self-report measure of PTSD symptoms that does not directly map onto *DSM-IV* criteria for PTSD. Future studies might adopt a self-report measure more closely tied to *DSM-IV* or an interview-based assessment. In addition, mediators or third variables may be operating differently according to the type of trauma, and future studies might productively explore whether mediators are similar across a variety of traumatic exposures.

Given the self-report methodology utilized in this study, it is important to acknowledge the concern surrounding the potential biasing impact of PTSD symptomatology, on reports of trauma exposure (King et al., 2000). Specifically, does PTSD influence whether reports of stressor exposure obtained at Time 1 (e.g., upon return from the Gulf War) are consistent (as opposed to increase or decrease) when assessed on a later occasion? King et al. (2000) employed sophisticated statistical techniques (e.g., cross-lagged panel design) to examine this question in this sample of Gulf War veterans. These authors concluded that the effects of PTSD symptomatology on increased reporting of stressor exposure over time are minimal (King et al., 2000). Clearly, however, future research replicating the finding that trauma exposure increases risk for subsequent trauma exposure utilizing more objective trauma criterion would be useful. Finally, although the majority of these effect sizes may be labeled as small effects (Cohen, 1988), we believe that these effects suggest a relation worthy of further investigation given the complexity of the phenomenon of interest (see, e.g., McClelland & Judd, 1993, for a discussion of smaller effect sizes and lower power in nonexperimental research) as well as the costs of trauma exposure.

This study demonstrated that men and women exposed to combat during the Gulf War are at increased risk of exposure to traumatic life events in the approximately 2 years following the Gulf War, and that this increased risk is partially mediated by the individual's level of PTSD symptomatology. These findings suggest that successful treatment of PTSD symptoms may also reduce risk of subsequent exposure to trauma. Future studies should examine in greater detail the process by which PTSD symptomatology confers increased risk for subsequent trauma exposure. If we are able to intervene in a cycle of repeated exposure to trauma for even a subset of individuals, the potential positive consequences (e.g., quality of life, financial costs) are enormous.

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